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1: J Cell Physiol. 1999 Sep;180(3):355-64.

Related Articles, Links

Clusterin (Apo J) regulates vascular smooth muscle cell differentiation in vitro.

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Previously we reported a significant and substantial increase in the synthesis and secretion of clusterin in cultured porcine vascular smooth muscle cells (VSMC) during the time when the VSMC culture modulates from a proliferating monolayer morphology to a nodular cell culture morphology. That in vitro process appears to recapitulate some aspects of in vivo vascular remodeling in response to injury and is facilitated by the presence of a well-developed extracellular matrix. To directly test the hypothesis that clusterin regulates VSMC phenotypic modulation, cultured VSMC were stably transfected with an expression plasmid containing the full-length murine clusterin sequence in antisense orientation. Twenty-four clones were selected on the basis of neomycin resistance and characterized for clusterin expression and culture morphology. In contrast to clone SM-CLU18AS, which expresses a high level of clusterin and forms multicellular nodules, clone SM-CLU13AS expresses a low level of clusterin and does not form nodules even in the presence of a preformed collagen gel. Importantly, clusterin-negative SM-CLU13AS retains the ability to form nodules in an environment containing exogenous clusterin. SM-CLU13AS forms nodules when cultured in Matrigel (which contains clusterin) and in the presence of clusterin-containing conditioned media prepared from nodular SMC cultures or SM-CLU18AS cultures. These results demonstrate that clusterin is required for VSMC nodule formation and suggest that it may play a role in smooth muscle cell reorganization in the vascular wall.

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